CHAPTER 63
Examination of the Reflexes

KEY TEACHING POINTS

- Abnormal muscle stretch reflexes *in isolation* do not necessarily indicate disease. Instead, muscle stretch reflexes are abnormal if any of the following findings are present: hyporeflexia is associated with additional signs of lower motor neuron disease (weakness, atrophy, fasciculations); hyperreflexia is associated with additional signs or upper motor neuron disease (weakness, spasticity, Babinski sign); the reflex response is asymmetric; or the reflex is unusually brisk when compared with a reflex from a higher spinal level.
- Criteria for the pathologic upgoing toe (Babinski response) include the following: the extensor hallucis longus muscle contracts; there are abnormal fine motor movements of the affected foot (e.g., abnormal foot tapping); other flexor muscles in the limb contract at least slightly (e.g., hamstrings, tensor fascia lata); and the response is reproducible.
- Primitive reflexes (e.g., palmomental reflex, glabellar reflex, grasp reflex, snout reflex, and suck reflex) are common findings in frontal lobe disease, parkinsonism, and dementing illnesses.

Reflexes are involuntary contractions of muscles, induced by specific stimuli. In the neurologic examination there are three types of reflexes: (1) muscle stretch reflexes (deep tendon or myotatic reflexes), (2) cutaneous reflexes, and (3) primitive reflexes (or release reflexes). This chapter also discusses the **Babinski response**, which is an abnormal cutaneous reflex of the foot that appears in upper motor neuron disease.

REFLEX HAMMERS

I. TYPES OF REFLEX HAMMERS

Early in the history of reflex testing*, clinicians used various implements to elicit reflexes; the great British neurologist Gowers used the ulnar aspect of his hand or his rigid stethoscope. Other clinicians were less selective, using paper weights, laboratory stands, or even table lamps.[2-4] In the late 1800s and early 1900s many different reflex hammers were produced, some of which remain popular nowadays.

A. TAYLOR HAMMER

The Taylor hammer was developed in 1888 by J.M. Taylor, personal assistant to S. Weir Mitchell at the Philadelphia Orthopedic Hospital and Infirmary for Nervous

*Reflex testing became common after Erb and Westphal simultaneously discovered the value of muscle stretch reflexes in 1875.[1]

Disease. This hammer has a tomahawk-shaped soft rubber hammer with a broad edge for percussing most tendons and a rounded point for reaching the biceps tendon or percussing muscles directly. The original handle ended in an open loop; the pointed end was added in approximately 1920 for use in eliciting cutaneous reflexes.[4]

B. QUEEN SQUARE HAMMER

The Queen Square hammer was developed by a Miss Wintle, head nurse at the National Hospital for Nervous Diseases at Queen's Square, London, who for years made hammers from ring pessaries, solid brass wheels, and bamboo rods to sell to resident medical officers. This hammer has a rubber-lined disc attached to the end of a long rod, like a wheel on an axle.[2]

C. BABINSKI HAMMER (BABINSKI/RABINER HAMMER)

This hammer has a handle that can be removed and attached either perpendicular or parallel to the disc-shaped head. Babinski's name probably reflects marketing more than innovation.[4]

D. TROEMNER HAMMER

The Troemner hammer, the only one of the four that actually resembles a hammer, was made popular in this country by the Mayo Clinic, where the neurologist Woltman introduced it in 1927.[5]

II. CLINICAL SIGNIFICANCE

No study has demonstrated any hammer to be superior to another, and selection depends more on personal preference and tradition. The Taylor is popular in America, the Queen Square in England, and the Troemner in continental Europe.[6] The built-in pins of some models (e.g., older Babinski hammers), designed for testing pain sensation and cutaneous reflexes, should not be used because they could transmit infections.[7]

MUSCLE STRETCH REFLEXES

I. DEFINITION

Muscle stretch reflexes are involuntary contractions of muscles induced by a brisk stretch of the muscle. Muscle stretch reflexes are usually named after the muscle being tested (Table 63.1), the one notable exception being the Achilles or ankle

TABLE 63.1 Common Muscle Stretch Reflexes[9-20]

Name of Reflex	Peripheral Nerve	Spinal Level
Brachioradialis	Radial	C5-C6
Biceps	Musculocutaneous	C5-C6
Triceps	Radial	C7-C8
Quadriceps (patellar)	Femoral	L2-L4
Medial hamstring*	Sciatic	L5, S1
Achilles (ankle)	Tibial	S1

*An online video demonstrating the medial hamstring reflex is available in reference[8].

jerk. Although these reflexes are often called **deep tendon reflexes**, this name is a misnomer because tendons have little to do with the response, other than being responsible for mechanically transmitting the sudden stretch from the reflex hammer to the muscle spindle. In addition, some muscles with stretch reflexes have no tendons (e.g., "jaw jerk" of the masseter muscle).

Most healthy persons have the muscle stretch reflexes listed in Table 63.1.

II. TECHNIQUE

A. METHOD

The usual stimulus is a sharp tap with the reflex hammer on the muscle's tendon, near where the tendon inserts distally on bone. The Achilles reflex is also elicited sometimes by the plantar strike method, in which the reflex hammer strikes the clinician's hand, which is resting on the ball of the foot. In clinical studies of the Achilles reflex, both the plantar strike and tendon strike methods are equivalent.[21-23]

B. GRADING REFLEX AMPLITUDE

The most important observation during reflex examination is the reflex's amplitude. Unlike examination of motor strength, examination of reflexes lacks a single universally accepted grading system. Proposed schemes range from S. Weir Mitchell's original four grades to the Mayo Clinic's nine grades.[24] A five-point grading system (i.e., grades 0 through 4), reproduced in Table 63.2, is recommended by the National Institute of Neurological Disorders and Stroke (NINDS).[25]

C. REINFORCEMENT: THE JENDRASSIK MANEUVER

According to the NINDS scale (see Table 63.2), grade 1 reflexes describe reflexes made conspicuous by reinforcement maneuvers, and grade 0 reflexes are those that are absent despite reinforcement. The most common method of reinforcing reflexes is the **Jendrassik maneuver**. In 1885 Erno Jendrassik reported that having the patient "hook together the flexed fingers of his right and left hands and pull them apart as strongly as possible" while the clinician taps on the tendon enhances the reflexes of normal patients.[2] Reflex enhancement with this maneuver persists as long as the patient is pulling apart the arms, up to 10 seconds in some studies.[26,27] In one study of normal elderly patients the absent ankle jerk was made to appear 70% of the time using reinforcing maneuvers.[28]

TABLE 63.2 NINDS[25]* Muscle Stretch Reflex Scale

Grade	Finding
0	Reflex absent
1	Reflex small, less than normal; includes a trace response or a response brought out only with reinforcement
2	Reflex in lower half of normal range
3	Reflex in upper half of normal range
4	Reflex enhanced, more than normal; includes clonus if present, which optionally can be noted in an added verbal description of the reflex

NINDS, National Institute of Neurological Disorders and Stroke, based upon reference 25.

III. CLINICAL SIGNIFICANCE

A. AMPLITUDE OF REFLEX

The amplitude of muscle stretch reflexes depends on the integrity of the lower and upper motor neurons innervating the reflex (see Fig. 61.2 in Chapter 61 for definition of lower and upper motor neurons). (1) The lower motor neurons of a reflex are its peripheral nerve (second column in Table 63.1) and its spinal segment (third column in Table 63.1): disease at either of these locations *reduces* or *abolishes* the relevant reflex. (2) The upper motor neurons are the descending corticospinal pathways innervating the reflex: disease anywhere along this pathway (e.g., cerebral hemisphere, brain stem) *exaggerates* the reflex. (3) Disease of the *spinal cord*, where both upper and lower motor neurons reside, abolishes the reflex *at* the level of the lesion (lower motor neuron response) and exaggerates all reflexes from spinal levels *below* the level of the lesion (upper motor neuron response).

Nonetheless, absent or exaggerated reflexes, by themselves, do not signify neurologic disease.[29-31] For example, 6% to 50% of elderly persons without neurologic disease lack the ankle jerk bilaterally, despite the Jendrassik maneuver,[28,32] and a small percentage of normal individuals have generalized hyperreflexia.[29-31,33] Instead, the absent or exaggerated reflex is significant only when it is associated with one of the following clinical settings:

1. The absent reflex is associated with other findings of lower motor neuron disease (weakness, atrophy, fasciculations).
2. The exaggerated reflex is associated with other findings of upper motor neuron disease (i.e., weakness, spasticity, Babinski sign).
3. The reflex amplitude is asymmetric, which suggests either lower motor neuron disease of the side with the diminished reflex or upper motor neuron disease of the side with exaggerated reflex.
4. The reflex is unusually brisk compared with reflexes from a higher spinal level, which raises the possibility of spinal cord disease at some level of the spinal cord between the segments with exaggerated reflexes and those with diminished ones.

B. LOCALIZING VALUE OF DIMINISHED REFLEXES

In patients with nerve complaints of the arm or leg suggesting disorders of the cervical or lumbosacral nerve roots, the diminished reflex has important localizing value that indicates a lesion of the reflex's respective spinal root (see Table 63.1). A diminished biceps or brachioradialis reflex indicates C6 radiculopathy (likelihood ratio [LR] = 14.2),[17] a diminished triceps reflex indicates C7 radiculopathy[†] (LR = 3),[17,34] a diminished quadriceps reflex indicates L3 or L4 radiculopathy (LR = 8.5),[35-38] a diminished medial hamstring reflex indicates L5 disease (LR = 6.2),[20] and a diminished Achilles reflex indicates S1 radiculopathy (though only modestly, LR = 2.7)[35-37,39-41] (see also Chapter 64).

C. ANKLE JERK AND DIABETIC PERIPHERAL NEUROPATHY

In one study of adult outpatients with type 2 diabetes mellitus the absent Achilles reflex detected peripheral neuropathy (defined by nerve conduction testing) with a sensitivity of 92%, specificity of 67%, positive LR = 2.8, and negative LR = 0.1.[42] This indicates that in diabetic patients, the *presence* of the ankle jerk greatly *decreases*

[†]C6 and C7 radiculopathies are much more common than C5 or C8 radiculopathies (see Chapter 64).

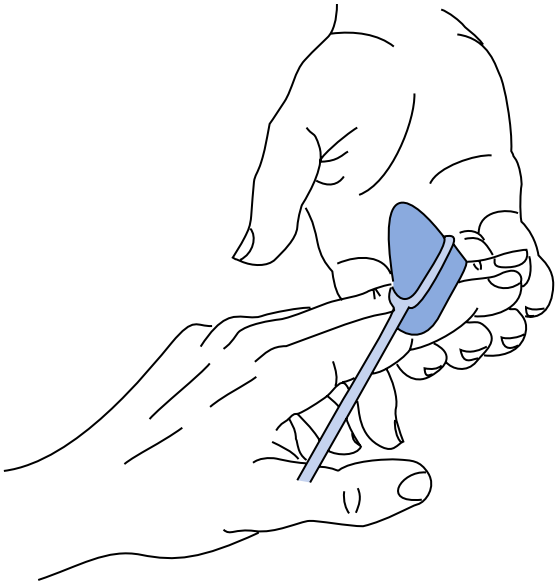

FIG. 63.1 FINGER FLEXION REFLEX. After positioning the patient's hand in the supinated position with fingers slightly flexed, the clinician places his own index and middle fingers across the tips of the patient's fingers and taps them with the reflex hammer. Reflex contraction of the patient's finger flexor muscles is a positive response.

probability of diabetic peripheral neuropathy (LR = 0.1). In this study, examination of the ankle jerk was more accurate than neuropathic symptoms, duration of diabetes, or retinopathy in predicting neuropathy.

D. ADDITIONAL FINDINGS IN THE HYPERREFLEXIC PATIENT

The physical finding of hyperreflexia has generated more eponyms in physical diagnosis than any other physical finding,[‡] even though the basic pathophysiology for all exaggerated reflexes is the same (i.e., loss of corticospinal inhibition) and the reflexes differ only by which muscle is stretched and which method the clinician uses to stretch the muscle. Of the many findings that have been described in hyperreflexic patients, commonly recognized ones are finger flexion reflexes, jaw jerks, clonus, and irradiating reflexes.

1. FINGER FLEXION REFLEXES

Finger flexion reflexes were introduced by Hoffman in approximately 1900. In a positive response, sudden stretching of the finger flexors causes the finger flexors to involuntarily contract (therefore the finger flexion reflex is no different from any other muscle stretch reflex). There are many ways to elicit this finding, each with its own eponym (e.g., **Hoffman sign**, **Rossolimo sign**, **Troemner sign**, **Bechterew reflex**). One of these methods is described in Fig. 63.1. Like other exaggerated reflexes, finger flexion reflexes by themselves have little diagnostic value (i.e., they

[‡] Dorland's Medical Dictionary lists 115 neurologic reflexes, 46 having eponyms.[43]

are detectable in 3% of healthy college students),[33] and, to be significant, they must accompany one of the settings described previously in the section on Amplitude of Reflex.

2. JAW JERK

The **jaw jerk** was originally described by Morris Lewis in 1882.[44,45] In a positive response, sudden stretching of the masseter muscle causes reflex contraction, moving the jaw briskly upward. With the patient's jaw slightly open, the clinician can elicit the reflex by tapping with a reflex hammer directly on the chin or on a tongue blade resting on the lower teeth or tongue. An exaggerated jaw jerk, sometimes appearing with clonus (see later), implies bilateral disease above the level of the pons (e.g., pseudobulbar palsy).[29] For example, in patients with spastic tetraparesis, an exaggerated jaw jerk excludes cervical cord disease and points to pyramidal tract disease above the pons.

3. CLONUS

Clonus is a self-sustained, oscillating stretch reflex induced when the clinician briskly stretches a hyperreflexic muscle and then continues to apply stretching force to that muscle. Each time the muscle relaxes from the previous reflex contraction, the applied stretching force renews the reflex, setting up a rhythmic series of muscle contractions that continue as long as the tension is applied. These rhythmic oscillations (clonus) are most easily elicited in the foot (usually with oscillations of 5 to 8 Hz), by briskly dorsiflexing the patient's ankle. Clonus also may be elicited in the quadriceps, finger flexors, jaw, and other muscles.

As expected mathematically, the frequency of clonus varies inversely with the length of the reflex path ($r = -0.80$, $p < 0.001$). Clonus of the wrist has a higher frequency than that of the ankle, simply because the nerves to the forearm are shorter than those to the calf.[46]

Instructive videos are available of clonus at the ankle,[47] wrist,[48] and jaw.[49]

4. IRRADIATION OF REFLEXES

In some hyperreflexic patients, the blow of the reflex hammer is conducted mechanically through bone and tissues, where it may stretch hyperexcitable muscles at distant sites, thus producing additional, unexpected movements (e.g., crossed adductor reflex).[30,50] In addition, if this distant irradiation of a reflex is combined with paralysis of the reflex of interest, paradoxical movements or *inverted* reflexes may appear.

A. CROSSED ADDUCTOR REFLEX

Tapping on the medial femoral condyle, patella, or patellar tendon causes the contralateral adductor muscle to contract, moving the contralateral knee medially.[51]

B. INVERTED SUPINATOR REFLEX

The **inverted supinator reflex** (the supinator reflex is the brachioradialis reflex) was introduced by Babinski in 1910. This sign indicates spinal cord disease at the C5 to C6 level.[30,52,53] In a positive response, tapping on the brachioradialis muscle fails to flex the elbow but instead flexes the fingers. The lesion at C5 to C6 eliminates the brachioradialis reflex (lower motor neuron) but exaggerates all reflexes below that level (upper motor neuron), including the finger flexion reflexes (C8), which are stimulated by mechanical conduction of the blow on the brachioradialis.

C. INVERTED KNEE JERK[54]

The **inverted knee jerk** indicates spinal cord disease at the L2 to L4 level. In the positive response, attempts to elicit the knee jerk instead cause paradoxical knee flexion. Its two components are denervation of L2 to L4 (thus paralyzing the quadriceps jerk) and conduction of the blow to the muscle spindles of the hamstrings (innervated by the L5 to S1 level and made hyperexcitable by the same lesion).

CUTANEOUS REFLEXES (SUPERFICIAL REFLEXES)

I. DEFINITION

Cutaneous reflexes are involuntary muscle contractions that follow stimulation of the skin surface by scratching, stroking, or pinching.

II. SUPERFICIAL ABDOMINAL REFLEX (T6 TO T11)

A. TECHNIQUE

In the superficial abdominal reflexes, stroking the skin of the abdomen causes the underlying abdominal wall muscle to contract, sometimes pulling the umbilicus towards the stimulus (see the reference by Gosavi[55] for an online video). The clinician usually tests one abdominal quadrant at a time using a side-to-side motion with a wooden applicator stick or the pointed end of the reflex hammer handle. The abdominal reflexes appear just as often whether the direction is medial to lateral or lateral to medial.[56]

B. CLINICAL SIGNIFICANCE

According to traditional teachings, superficial abdominal reflexes disappear with both upper and lower motor neuron disease. However, their clinical value is slight because they are also absent in approximately 20% of normal individuals, more so in the elderly.[56,57] Moreover, the observation of asymmetric reflexes or ones preserved only in the upper quadrants, patterns traditionally associated with neurologic disease, also are a common finding in healthy persons.[56-58]

III. BULBOCAVERNOSUS REFLEX (S2 TO S4)

A. TECHNIQUE

After positioning the patient in the lithotomy position, sudden manual compression of the glans penis or clitoris causes reflex contraction of the bulbocavernosus muscle and external anal sphincter. The reflex is detected either by palpating the skin behind the scrotum (bulbocavernosus muscle) or, more commonly, placing the index finger in the anal canal (external anal sphincter). Other effective stimuli are percussing the suprapubic area[59] or pulling the retention balloon of an indwelling Foley catheter against the bladder neck.[60]

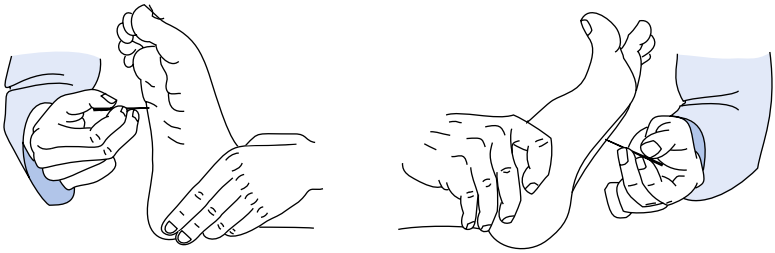

FIG. 63.2 BABINSKI RESPONSE. Drawing of the normal plantar cutaneous reflex (*left*) and the Babinski response (*right*), adapted from photographs taken by Babinski himself in 1900.[63]

B. CLINICAL SIGNIFICANCE

The bulbocavernosus reflex is one of the few ways to test the conus medullaris (distal end of the spinal cord) and the S2 to S4 pelvic nerves (the only other bedside test of this region is testing sensation in the perineal, or "saddle," area).[60-62] This reflex is particularly important in patients with urinary retention, which may be caused by disease of the pelvic nerves or cauda equina. In one study of consecutive patients referred for urodynamic studies,[60] most of whom had difficulty with urination, an *absent* reflex predicted disease in the S2 to S4 segments only modestly in women (LR = 2.7) but much better in men (LR = 13). The modest accuracy of the sign in women may reflect damage to the pudendal nerve from prior childbirth or pelvic surgery.[60] In this study the *presence* of a bulbocavernosus reflex was unhelpful; although the positive response is expected in patients with urinary retention from common disorders like prostate hypertrophy, it also is commonly found in incomplete lesions of the sacral nerves.

In spinal cord injury above the S2 to S4 level (i.e., lesion of upper motor neurons innervating the S2 to S4 segment), the bulbocavernosus reflex also disappears, but only temporarily for a period of 1 to 6 weeks.[60]

BABINSKI RESPONSE

I. DEFINITION

The Babinski response is an abnormal cutaneous reflex found in upper motor neuron disease affecting the muscles of the foot. In these patients, scratching the sole of the patient's foot causes an upward movement of the great toe, instead of the normal downward movement (Fig. 63.2). Much revered and researched, this reflex was originally described by Babinski in 1896.[63,64] It goes by various names, including Babinski response, Babinski sign, Babinski reflex, upgoing toe, and extensor response.

In some patients with bilateral corticospinal tract disease, scratching the foot may even cause the contralateral great toe to move upward, a response termed **crossed dorsiflexion** or **crossed extensor response.**[63]

II. PATHOGENESIS

In response to painful stimuli applied to the lower limbs, most mammals rapidly withdraw that limb by flexing the hips and knees and dorsiflexing the feet and toes.

This primitive reflex, the **flexion response**, also occurs in human infants until the age of 1 or 2 years, after which the developing pyramidal tracts cause two important changes: (1) the flexion response becomes less brisk, and (2) the toes no longer move upward but instead move downward because of the interval development of a normal plantar cutaneous reflex.[65] If pyramidal tract disease develops later in the person's life, the normal plantar cutaneous reflex disappears, and, instead, painful stimulation of the foot causes the great toe to again move upward.

The use of the term *extensor response* to describe the Babinski response is unfortunate and confusing: even though anatomists have always named the upward movement "extension" (e.g., great toe extensor muscle), physiologists have named the same upward movement "flexion" (e.g., the primitive flexion response discussed earlier).

III. TECHNIQUE

A. ELICITING THE RESPONSE

Of the many ways to elicit this reflex,[66] a slow (i.e., 5 to 6 seconds) hockey-stick stroke beginning on the lateral plantar surface is best, using a wooden applicator stick, key, or pointed handle of the reflex hammer (see Fig. 63.2). This method is superior to other methods, including scratching the lateral sole, scratching below the lateral malleolus (**Chaddock method**), rubbing the anterior shin (**Oppenheim method**) or—the least effective stimulus—squeezing the calf (**Gordon method**).[67,68]

B. INTERPRETING THE RESPONSE

Helpful guidelines in assessing an equivocal toe response, based on careful electrodiagnostic studies and patient follow-up, are as follows: (1) The pathologic upgoing toe results from contraction of the extensor hallucis longus muscle, whose tendon is conspicuous under the skin on top of the great toe.[69,70] Movement of the toe per se is not critical and may be prevented by joint disease. Moreover, the toe may seem to be upgoing—without contraction of the extensor hallucis longus muscle—when the ankle dorsiflexes or when the toe returns from an initial downward movement. (2) More than 90% of the time, the foot with the pathologic upgoing toe is weak or has difficulty with fine motor movements. An excellent test of fine motor movement is rapid foot tapping against the examiner's hand: normal persons accomplish 20 to 40 taps per 10 seconds. (3) The pathologic upgoing toe coincides with a flexion response in the whole limb, which may be slight but is evident in the ipsilateral tensor fascia lata and hamstrings. (4) The pathologic upgoing toe is reproducible.[65,71,72]

As Babinski himself pointed out, fanning of the toes is a normal phenomenon and not part of the pathologic response.[63,65]

IV. CLINICAL SIGNIFICANCE

A. ASSOCIATED CONDITIONS

The Babinski response is found in both destructive lesions of the pyramidal tracts (see Chapter 61) and in many metabolic disorders affecting these tracts, most of which are associated with altered mental status, such as seizures, meningitis, drug overdose, and renal and hepatic failure.[63] In patients with a variety of neurologic

complaints who undergo neuroimaging, the Babinski response greatly increases the probability of a lesion in the contralateral cerebral hemisphere (LR = 8.5, see Chapter 61).

B. FALSE-NEGATIVE RESPONSE

Patients may have pyramidal tract disease yet lack the upgoing toe (i.e., false-negative response) because they have the following: (1) spinal shock,[69] (2) a peroneal palsy denervating the muscles that dorsiflex the great toe (a common problem in bedridden patients due to pressure against the head of the fibula),[69] or (3) pyramidal tract disease sparing the muscles of the foot (e.g., upper motor neuron weakness that is confined to the arm of that side).[73]

PRIMITIVE REFLEXES

I. DEFINITION

Primitive reflexes (or **release reflexes**) are a hodgepodge of reflexes that are present normally in infants but disappear during normal development of the central nervous system, only to reappear sometimes later in life when neurologic disease or aging removes (or "releases") the inhibiting influences of the central nervous system.[74] Among many primitive reflexes,[75] the more common ones are the palmomental reflex, glabellar reflex, grasp reflex, snout reflex, and suck reflex.

II. TECHNIQUE

A. PALMOMENTAL REFLEX

In this reflex a key or other blunt object is used to apply an unpleasant stimulus to the patient's thenar eminence, stroking it briskly in a proximal to distal direction. A positive response is a brief contraction of the ipsilateral mentalis muscle, causing the ipsilateral lower lip to protrude, rise, or wrinkle.[76] An instructive video of the finding is available.[77]

The wrinkle response at the corner of the mouth is probably the beginnings of a wince that would develop with more painful stimuli.[78] Theoretically the stimulus could be applied anywhere on the skin of the patient's body, and indeed, descriptions of similar response after stimulation of the patient's arm, chest, trunk, sole of the foot, and tongue have all appeared.[78] The most sensitive area, however, is the thenar eminence.[79]

Marinesco and Radovici discovered the palmomental reflex in 1920.[78]

B. GLABELLAR REFLEX

The stimulus for the glabellar reflex is light taps with the finger or soft rubber reflex hammer, approximately two times per second, over the patient's glabella. Although most normal persons respond to this by blinking bilaterally, the blinking stops after the first few taps in normal individuals. Persistent blinking is a positive response, although there is no consensus whether habituation should be indefinite or just beyond a certain number of blinks (e.g., more than four successive blinks).

The glabellar reflex is sometimes called the **blink reflex** or **Myerson reflex**, although the original description was by Overend in 1896.[80]

C. GRASP REFLEX

In the grasp reflex the clinician places his index and middle fingers over the thenar aspect of the patient's wrist and exerts pressure on the skin while withdrawing the fingers between the patient's thumb and index finger. In a positive response the patient grasps the clinician's fingers, and the grasp progressively increases as the clinician attempts to withdraw.[75]

III. CLINICAL SIGNIFICANCE

A. GENERAL COMMENTS

Primitive reflexes are common findings in frontal lobe disease,[81] parkinsonism,[82-85] dementing illnesses,[86-90] and advanced human immunodeficiency virus (HIV) infection.[91] Other than the grasp reflex (see later), the precise neuroanatomic cause of these reflexes is unknown.

B. PALMOMENTAL REFLEX

The palmomental reflex is bilateral 38% to 75% of the time and unilateral 25% to 62% of the time.[92,93] The side of the reflex does not correlate with the side of the lesion.[76,92] In one study of 39 patients with a unilateral palmomental reflex, 44% had an ipsilateral cerebral hemispheric lesion, 36% a contralateral lesion, 10% bilateral lesions, and 10% no lesions.[93] In patients with Parkinson disease, the palmomental reflex correlates with the degree of akinesia, and the reflex often disappears with the onset of levodopa-induced dyskinesias.[82]

C. GLABELLAR REFLEX

The afferent limb of the glabellar reflex is the trigeminal nerve, and the efferent limb is the facial nerve. Lesions of either nerve may interrupt the reflex (although in facial nerve palsy, the blinking continues on the sound side). This reflex is also a common finding in Parkinson disease, and in these patients the positive response may reverse after administration of levodopa.[83]

D. GRASP REFLEX

A positive grasp reflex is common in frontal lobe disease, and, if both arms can be tested (i.e., no paralysis), the grasp reflex when present is usually bilateral.[81] In patients with dementia the sign correlates with more severe cognitive and functional impairment and greater loss of pyramidal cells in the frontal lobe.[86,87,90] Among patients admitted to a neurologic ward, a positive grasp reflex (defined as no habituation with three successive strokes) predicted discrete lesions in the frontal lobe or deep nuclei and subcortical white matter with a sensitivity of 13% to 50%, specificity of 99%, and positive LR of 19.1.[81,94]

E. PRIMITIVE REFLEXES AND NORMAL AGING

The palmomental and glabellar reflexes, but not the grasp reflex, may appear in normal persons, although the reported frequencies from different studies vary widely.[88,89,91,95] The reported frequency for the palmomental sign in normal persons varies from 3% to 70%, and that for the glabellar sign, from 3% to 33%.[82,89,91,95-98] A few of these "normal" persons with primitive reflexes undoubtedly have subclinical disease, as indicated by lesions in the basal ganglia or subcortical white matter on magnetic resonance imaging (MRI).[96] However, others have no evidence of neurologic disease, although importantly, their findings differ

from the pathologic response in two important ways: (1) the primitive reflex of patients without neurologic lesions is weak and fatiguable, disappearing after the first few repetitive stimuli spaced evenly apart,[74] and (2) the primitive reflex of patients without neurologic lesions is an isolated finding. For example, less than 1% of normal persons have a positive palmomental reflex if it is defined as persistence beyond five or more strokes of the thenar eminence.[79,82] In addition, even if the definition of a positive response includes fatigable primitive reflexes, less than 12% of normal persons have two primitive reflexes, and less than 2% have three or more primitive reflexes.[91,95,97-99]

The references for this chapter can be found on www.expertconsult.com.

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